

# Unusual Presentation of a Rare Case of Nonketotic Hyperglycemia with Brain Parenchymal Findings

Dhawal Kaushal<sup>1</sup>, Manisha Joshi<sup>2</sup>

**Abstract:** *Glucose is essential for normal functioning of the brain however the extreme (high or low) levels in blood can lead to various complications. We hereby present a case of non-ketotic hyperglycaemia ((NKH) who presented to us with history of long term uncontrolled diabetes. The diagnosis was not straight forward however the atypical imaging findings can suggest the diagnosis if the radiologist is aware of its variable presentations. Our patient showed postictal cortical areas of hyperintensities and subcortical hypointensities involving the left parieto-temporo-occipital region attributed to cell edema caused by NKH and seizures, but however were reversible when hyperglycemia and seizures were well managed by hypoglycaemic agents and anticonvulsants.*

**Keywords:** Non Ketotic Hyperglycemia (NKH), Cortical hyperintensities, Subcortical hypointensities

## 1. Introduction

### Literature Survey

### Case Report

We present a 51yr old lady who is a known case of diabetes mellitus, rheumatoid arthritis, sjogren syndrome with sensory ganglionopathy came with chief complaints of high grade fever with headache for 20days, fluctuating behaviour for 18 days, visual hallucinations for 15days, with 2 episodes of convulsion 6 days back.

### On Neurological Examination

Cranial nerve examination was normal. Only finding that could be appreciated was right sided hemianopia. Reflexes, sensory examination and cerebellar signs were unremarkable.

**Hematological/ Biochemical laboratory reports** showed Leucocyte count- 5600, Hb- 10.8gm/dl, Platelet- 1.3 lakhs, ESR- 59, CRP- 71, Fever profile- Negative.

### RBS-472, HbA1c- 15

Thyroid, liver and renal profiles were normal.

### Urine Routine Examination

Occasional pus cells, ketones trace.

### CSF Routine microscopy

Proteins- 63mg/dl, Glucose- 202mg/dl, Total cell count- 1, ZN and Gram Stain- Negative.

**EEG** was suggestive of bilateral (L>R) parieto-occipital dysfunction.

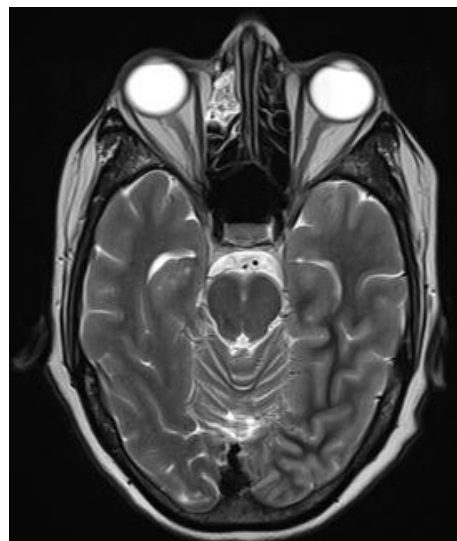
### Examination

Patient was conscious, confused with reduced attention span with speech spontaneity.

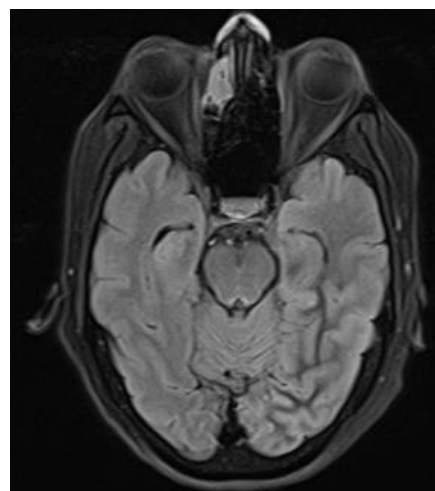
### MRI Brain

(Plain with contrast) was done which showed areas of gyriform thickening and abnormal T2/T2FLAIR hyperintensities seen involving the cortex of left parieto-temporo-occipital region showing evidence of restricted diffusion on DWI/ADC images with no evidence of blooming on GRE. These areas showed enhancement on

post contrast imaging. There was also an area of abnormal T2/T2FLAIR hypointensities seen involving the subcortical white matter of left parieto-temporo-occipital region.



**1(a)** Axial T2W image shows areas of gyriform thickening and abnormal T2/T2FLAIR hyperintensities seen involving the cortex of left parieto-temporo-occipital region. Areas of abnormal T2/T2FLAIR hypointensities seen involving the subcortical white matter of left parieto-temporo-occipital region.



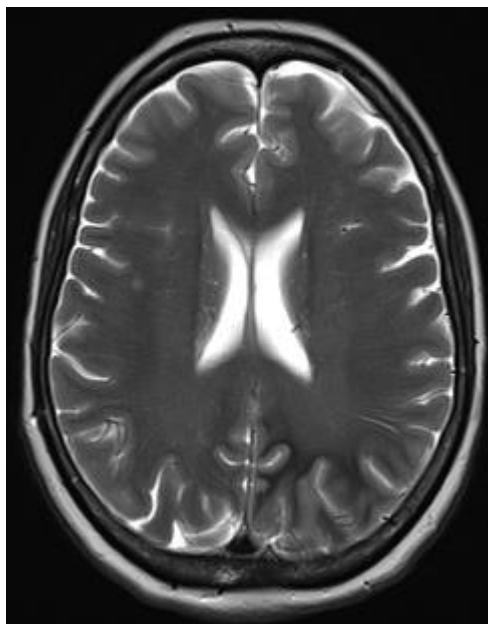
**(b)** Axial T2FLAIR image shows areas of gyriform thickening and abnormal T2/T2FLAIR hyperintensities seen involving the cortex of left parieto-temporo-occipital region. Areas

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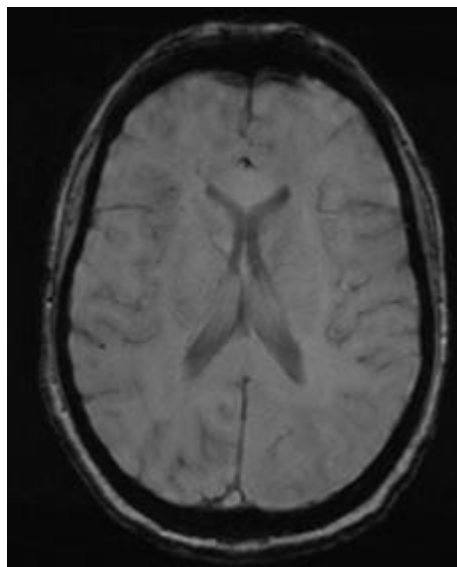
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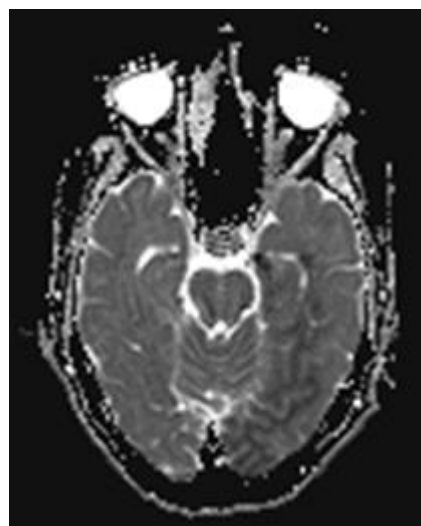
of abnormal T2/T2FLAIR hypointensities seen involving the subcortical white matter of left temporo-occipital region.



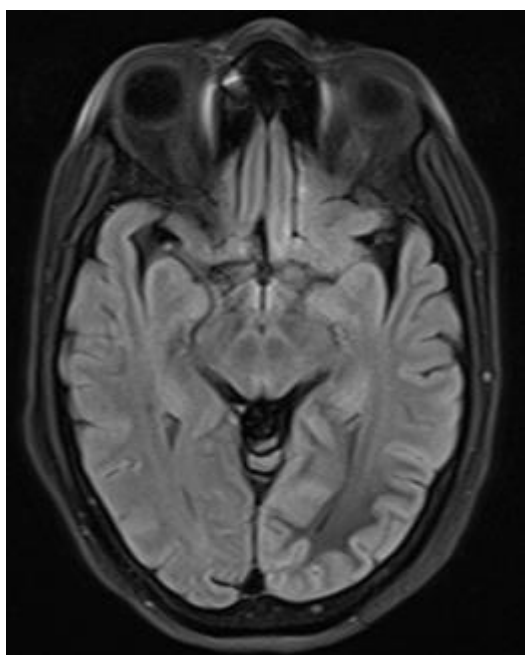
1(c) Axial T2W image shows areas of gyriform thickening and abnormal T2/T2FLAIR hyperintensities seen involving the cortex of left parietal region. Areas of abnormal T2/T2FLAIR hypointensities seen involving the subcortical white matter of left parietal region.



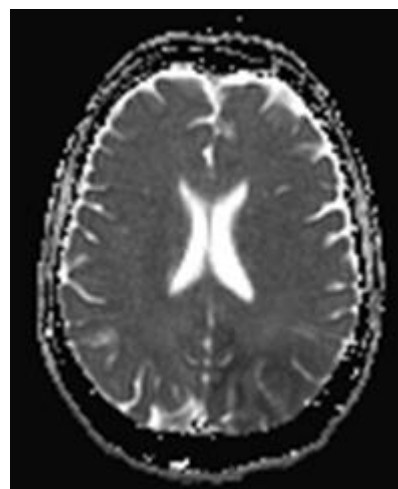
1(e) Axial GRE Image shows no evidence of blooming noted.



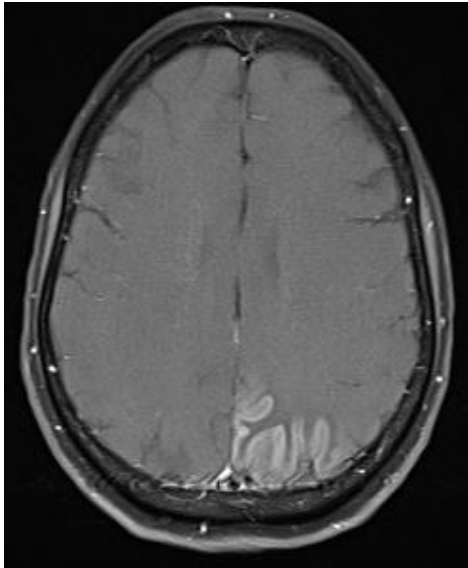
1(f) Low ADC values are noted in left temporo-occipital region suggesting restricted diffusion.



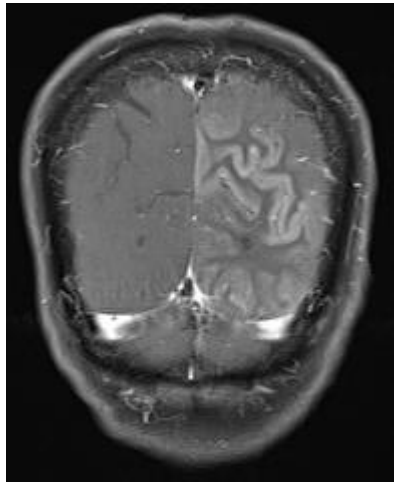
1(d) Axial T2FLAIR image shows areas of gyriform thickening and abnormal T2/T2FLAIR hyperintensities seen involving the cortex of left parieto-temporo-occipital region. Areas of abnormal T2/T2FLAIR hypointensities seen involving the subcortical white matter of left temporo-occipital region.



1(g) Low ADC values are noted in left temporo-occipital region suggesting restricted diffusion.



1(h) Axial Post contrast images showed evidence of enhancement.



1(i) Coronal Post contrast images showed evidence of enhancement.

## Differentials

### Hyperglycemic non ketotic brain parenchymal changes. Meningoencephalitis.

#### Outcome and Follow Up

Patient was managed conservatively and showed good neurological recovery. Diabetes was controlled initially with insulin and later with oral hypoglycaemic agents.

## 2. Results/ Discussion

Variations in the level of plasma glucose levels are commonly seen and can present with variable imaging findings. The patient can present with nonspecific, confusion, loss of memory, seizures, hemiparesis and sometimes even mimic a stroke. Non-ketotic hyperglycemia (NKH) as a cause of focal seizures is well described.[1] In some cases, partial seizures are the presenting feature of undiagnosed diabetes mellitus. In patients with NKH, focal motor seizures and partial epilepsy partialis continua are most commonly seen. The occipital seizures may have a variety of visual symptoms like blurring of vision, flickering of lights and objects and hemianopia as seen in our patient.[2] Occipital seizures have been found to be

associated with state of hyperglycemia with blood glucose levels ranging from 200 mg/dl to over 500 mg/dl. HbA1c has been mentioned in only a few cases and is found to be high, similar to our case (HbA1c) suggesting poor long-term glycemic control. Hence, it has been suggested that occipital seizures are attributed to long-term of poor control of diabetes as compared to an acute episode of hyperglycemia. [3]

The exact pathophysiology of seizures in hyperglycemia with lack of ketones(NKH) is not exactly known. There have been few hypotheses: 1) hyperosmolality gradient resulting in fluid shift causing intracellular dehydration triggering seizures. The mechanism postulated is cellular dehydration caused by hyperosmolality which results in inhibition of Krebs cycle causing compensatory increase in metabolism of GABA to succinic acid, these results in depletion of GABA which creates a hyperexcitable neuronal state. The explanation does not fully account for the cause of seizures as it may occur in hyperglycaemic state in the absence of significant hyperosmolality. Hyperglycemia itself may have a seizure triggering effect has also been suggested. Recently, hyperglycemia-related seizures have also been attributed to KATP channels.[2]

Subcortical hypointensities have been recently identified in relation to NKH and has also been attributed to transient accumulation of free radical during excitotoxic damage from seizure activity.

## 3. Conclusion

Subcortical T2 hypointensity is seen rarely and can be seen in patients with ischemia, multiple sclerosis, leptomeningeal metastasis and meningoencephalitis. In our patient, areas of gyriform thickening and abnormal T2/T2FLAIR hyperintensities seen involving the cortex of left parieto-temporo-occipital region showing evidence of restricted diffusion on DWI/ADC images demonstrating post contrast enhancement. The areas of abnormal T2/T2FLAIR hypointensities seen involving the subcortical white matter of left parieto-temporo-occipital region were also observed several days after NKH with focal seizures, hence, likely reflecting the cell edema. The radiologist should be well aware of the clinical, typical and atypical imaging appearances of various metabolic states which may assist in identifying the cause, making early diagnosis and prompt management so as to decrease the associated morbidity and mortality.

## 4. Learning Points

- Uncontrolled hyperglycemia can lead to changes in the brain parenchyma which can present new neurological findings.
- Blood sugar levels with HbA1C levels should be measured to assess the control of diabetes.
- Resolution of hyperglycaemic state can result in reversal of the brain parenchymal changes.

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## Author Profile



**Dr Dhawal Kaushal** has approximately 4 years of experience post M.D. He has worked in various hospitals in Chennai and Mumbai with few publications, posters and fellowships to his credit.



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